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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/768,744	02/02/2004	Christopher Hunter	25927	4909

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NATH & ASSOCIATES
112 South West Street
Alexandria, VA 22314

EXAMINER	
WOODWARD, CHERIE MICHELLE	
ART UNIT	**PAPER NUMBER**
1647	

DATE MAILED: 11/29/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/768,744	HUNTER ET AL.	
	Examiner	Art Unit	
	Cherie M. Woodward	1647	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *08 September 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-73* is/are pending in the application.

4a) Of the above claim(s) *27-72* is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-26 and 73* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☒ The specification is objected to by the Examiner.

10)☒ The drawing(s) filed on *02 February 2004* is/are: a)☒ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *9/8/06*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

1. Applicant's election with traverse of Group I (claims 1-5) in the reply filed on 8 September 2006 is acknowledged. The traversal is on the grounds that the independent claims of Groups I-IV are closely related, differing only in the targeted patient group for administration of an agent that increases WSX-1 activity. Applicant argues that Groups I-IV (claims 1-26 and new claim 73) can be searched together without undue burden on the Examiner. Applicant also traverses the requirement to elect one immune-related disorder from claim 20 if Group IV is elected. Applicant opines at length as to the additional restriction requirement over claim 20. Applicant's arguments have been fully considered but they are not persuasive.

Although Applicant elects Group I (claims 1-5), Applicant has amended the claims such that Group I now reads on Group IV, thus, it appears that Applicant has really elected Group IV, by amendment. The election will be treated as an election by original presentation.

However, in order to facilitate compact prosecution, the Examiner will examine amended claims 1-26 and 73, and the secondary restriction requirement will be withdrawn. As such, Applicant's arguments are moot.

The amended restriction groups are set forth as:

Group I, claims 1-26 and 73, drawn to a method of preventing or treating an immune disorder comprising administering an effective amount of an IL-27R/WSX-1 ligand.

Group II, claims 27-32, drawn to a pharmaceutical composition comprising an effective amount of an IL-27/WSX-1 ligand and a pharmaceutical carrier.

Group III, claims 33-72, drawn to a method of treating immune hyperreactivity comprising administering an effective amount of an agent that increases WSX-1 activity.

Formal Matters

2. Claims 1-73 are pending. Claims 27-72 are withdrawn as being drawn to non-elected inventions. Claims 1-26 and 73 are under examination.

Information Disclosure Statement

3. The information disclosure statement (IDS) submitted on 8 September 2006 has been considered. A signed copy is attached hereto.

Priority

4. Applicant has listed a priority document as "PCT/Not yet Assigned", filed 30 January 2004, in the Oath/Declaration of the instant application. However, the case number of that PCT document does not appear anywhere in the instant record and, accordingly, benefit to the international filing date of the PCT is unable to be accorded where the document cannot be located.

If applicant desires to claim the benefit of a prior-filed application under 35 U.S.C. 119(e), a specific reference to the prior-filed application in compliance with 37 CFR 1.78(a) must be included in the first sentence(s) of the specification following the title or in an application data sheet. For benefit claims under 35 U.S.C. 120, 121 or 365(c), the reference must include the relationship (i.e., continuation, divisional, or continuation-in-part) of the applications.

If the instant application is a utility or plant application filed under 35 U.S.C. 111(a) on or after November 29, 2000, the specific reference must be submitted during the pendency of the application and within the later of four months from the actual filing date of the application or sixteen months from the filing date of the prior application. If the application is a utility or plant application which entered the national stage from an international application filed on or after November 29, 2000, after compliance with 35 U.S.C. 371, the specific reference must be submitted during the pendency of the application and within the later of four months from the date on which the national stage commenced under 35 U.S.C. 371(b) or (f) or sixteen months from the filing date of the prior application. See 37 CFR 1.78(a)(2)(ii) and (a)(5)(ii). This time period is not extendable and a failure to submit the reference required by 35 U.S.C. 119(e) and/or 120, where applicable, within this time period is considered a waiver of any benefit of such prior application(s) under 35 U.S.C. 119(e), 120, 121 and 365(c). A benefit claim filed after the required time period may be accepted if it is accompanied by a grantable petition to accept an unintentionally delayed benefit claim under 35 U.S.C. 119(e), 120, 121 and 365(c). The petition must be accompanied by (1) the reference required by 35 U.S.C. 120 or 119(e) and 37 CFR 1.78(a)(2) or (a)(5) to the prior application (unless previously submitted), (2) a surcharge under 37 CFR 1.17(t), and (3) a statement that the entire delay between the date the claim was due under 37 CFR 1.78(a)(2) or (a)(5) and the date the claim was filed was unintentional. The Director may require additional information where

there is a question whether the delay was unintentional. The petition should be addressed to: Mail Stop Petition, Commissioner for Patents, P.O. Box 1450, Alexandria, Virginia 22313-1450.

If the reference to the prior application was previously submitted within the time period set forth in 37 CFR 1.78(a), but not in the first sentence(s) of the specification or an application data sheet (ADS) as required by 37 CFR 1.78(a) (e.g., if the reference was submitted in an oath or declaration or the application transmittal letter), and the information concerning the benefit claim was recognized by the Office as shown by its inclusion on the first filing receipt, the petition under 37 CFR 1.78(a) and the surcharge under 37 CFR 1.17(t) are not required. Applicant is still required to submit the reference in compliance with 37 CFR 1.78(a) by filing an amendment to the first sentence(s) of the specification or an ADS. See MPEP § 201.11.

5. Applicant has claimed priority to 60/444,494 (1/31/2003) and 60/519,074 (filed 10 November 2003). The filing date of the instant application is 2 February 2004. As such, the instant filing date occurred more than one year after the provisional filing of 60/444,494. Therefore, benefit is denied to 60/444,494. Additionally, the 60/519,074 provisional document is a copy of a publication, published in November 2003. The scope of the publication fails to correspond to the breadth of the instant claims. As such, benefit to the 60/519,074 provisional document is denied. Applicant is therefore accorded the benefit only to the filing date of the instant application, that of 2 February 2004.

Objections – Specification

6. The disclosure is objected to because of the following informalities: there are blocks in the text on page 106 that appear to be a typographical error. It appears that a centigrade symbol should have appeared where box symbols are presently located. Appropriate correction is required.

7. The lengthy specification has not been checked to the extent necessary to determine the presence of all possible minor errors. Applicant's cooperation is requested in correcting any errors of which applicant may become aware in the specification.

8. The use of the trademarks SIGMA, COSTAR, TRIZOL (p. 106), CHRION, BD PHARMINGEN, TWEEN-20 (p. 108), IMMULON IV, TWEEN, CARNOY'S SOLUTION, TOLUIDINE BLUE, SCHIFF'S (p. 110) have been noted in this application. They should be capitalized wherever they appear and be accompanied by the generic terminology.

Although the use of trademarks is permissible in patent applications, the proprietary nature of the marks should be respected and every effort made to prevent their use in any manner which might adversely affect their validity as trademarks.

Claim Objections

9. Claims 1-26 and 73 are objected to because of the following informalities: the specification refers to the ligand of interest as IL-27/WSX-1 throughout most of the specification and all of the examples. The ligand of interest is recited as IL-27R/WSX-1 in one paragraph on page 1 of the disclosure, but is primarily identified as IL-27/WSX-1 throughout the remainder of the disclosure. However, the claims recite the ligand of interest as IL-27R/WSX-1. IL-27 is considered the ligand of the IL-27 receptor (IL-27R). WSX-1 is known to be one of the two subunits of the IL-27R, the other being gp130. For purposes of compact prosecution, the designations of either IL-27/WSX-1 or IL-27R/WSX-1 will be read as referring to the WSX-1 portion of the IL-27R subunit of the IL-27 ligand/receptor complex. Appropriate correction/clarification is required.

10. Claims 21, 23, and 73 are objected to because of the following informalities: the disorder "amyotrophic lateral sclerosis" is misspelled in line 4 of each of the claims. Appropriate correction is required

Claim Rejections - 35 USC § 112, First Paragraph

Enablement

11. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

12. Claims 1-26 and 73 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claims contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The factors considered when determining if the disclosure satisfies the enablement requirement and whether any necessary experimentation is undue include, but are not limited to: 1) nature of the invention, 2) state of the prior art, 3) relative skill of those in the art, 4) level of predictability, 5) existence

of working samples, 6) breadth of claims, 7) amount of direction or guidance by the inventor, and 8) quantity of experimentation needed to make or use the invention. *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988).

The claims recite a method of preventing or treating an immune disorder in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said treating or preventing comprises immune suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the recited group; wherein said treating or preventing comprises immune activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27R/WSX-1 binding affinity or an antagonist antibody that suppresses IL-27R/WSX-1 activity; a method for modulating a T-helper cell mediated immune response in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL-27, and an agonistic antibody to IL-27R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27/WSX-1 binding activity or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; wherein the T-helper cell is Th1; wherein the T-helper cell is Th2; a method for modulating an interferon-γ mediated immune response in a patient in need thereof which comprises administering to a patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL027, and an agonistic antibody to IL027R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an active IL27 fragment which retains IL-27R/WSX-1 binding affinity, or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; a method for treating immune hyperreactivity in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; a method for treating an immune hyperreactivity disorder in a patient in need thereof, which comprises administering to said patient an effective amount of a IL-27R/WSX-1 ligand; wherein said immune disorder is selected from the group consisting of autoimmune disorders, hypersensitivity disorders, allergies, and asthma; wherein said immune disorder is selected from the recited group; a method for treating a T-helper cell mediated disorder in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein the T-helper cell mediated disorder is selected from the

recited group; a method for modulating a T-helper cell mediated autoimmune response in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said T-helper cell is Th1; wherein said T-helper cell is Th2; the method of claim1 wherein the disorder is selected from the recited group.

The specification does not reasonably provide enablement for prevention of any immune disorder in any species by any means. The skilled artisan cannot envision the prevention of an immune disorder. Prevention involves "attacking" the underlying cause of disease; i.e., disrupting the mechanisms which give rise to any given immune disorder. The skilled artisan is aware that the causes of immune disorders were unknown at the time of the invention herein. For purposes of enablement, the specification must provide reasonable detail in order for those skilled in the art to carry out the invention. In this case, the specification must disclose a means of preventing immune disorders regardless of the underlying causes of the immune disorders. The teachings of the specification do not enabled a person of ordinary kill in the art to make and use the claimed method of prevention. Moreover, "[p]atent protection is granted only in return for an enabling disclosure of an invention, not for vague intimations of general ideas that may or may not be workable." Genentech Inc. v. Novo Nordisk A/S, 108 F.3d at 1366, 42 USPQ2d at 1005 (Fed. Cir.), cert. denied, 118 S. Ct. 397 (1997), ("Tossing out the mere germ of an idea does not constitute an enabling disclosure").

Due to the large quantity of experimentation necessary to determine the etiology of any the claimed immune disorders such that it can be determined whether they can be prevented, the lack of direction/guidance presented in the specification regarding same, the absence of sufficient working examples directed to same, the complex nature of the invention, the state of the prior art establishing that the etiology of immune disorders is unknown, and the breadth of the claims, undue experimentation would be required of the skilled artisan to make and/or use the claimed invention.

Claim Rejections - 35 USC § 112, First Paragraph

Scope of Enablement

13. The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

14. Claims 1-26 and 73 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of assessing the role of IL-27/WSX-1 *in vitro,* in the development and

regulation of resistance to *Toxoplasma gondii* in WSX-1 deficient mice, does not reasonably provide enablement for a method of preventing or treating an immune disorder, a method of modulating a T-helper cell mediated immune response, a method for modulating an interferon-γ mediated immune response, a method for treating immune hyperreactivity in a patient, or a method for treating an immune hyperreactivity disorder in a patient by administering an antagonist or an agonist to suppress or activate the immune system of the patient. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make or use the invention commensurate in scope with these claims.

The factors considered when determining if the disclosure satisfies the enablement requirement and whether any necessary experimentation is undue include, but are not limited to: 1) nature of the invention, 2) state of the prior art, 3) relative skill of those in the art, 4) level of predictability, 5) existence of working samples, 6) breadth of claims, 7) amount of direction or guidance by the inventor, and 8) quantity of experimentation needed to make or use the invention. *In re Wands*, 858 F.2d 731, 737, 8 USPQ2d 1400, 1404 (Fed. Cir. 1988).

The claims recite as stated *supra*. The nature of the invention is drawn to a method of treating, preventing, or modulating an immune disorder, T-helper cell mediated response, interferon-γ mediated immune response, or hyperactivity disorder in a patient by administering an agonist or antagonist which suppresses or enhances WSX-1 activity, thereby suppressing or activating the immune system of the patient.

The state of the art discloses that p28 is a subunit of the IL-27 ligand, along with EBI3 (IL-27p28/EBV-induced gene 3). One of the subunits comprising the IL-27 receptor is WSX-1 (also known as TCCR or WSX-1/TCCR). WSX-1/TCCR is one of the two IL-27 receptor subunits, the other being gp130 (WSX-1/gp130) (see Timans *et al.*, US 2002/0164609 A1, published 7 November 2002, especially at p. 2, column 1). It is well known in the art that the heterodimeric IL-27 receptor (comprised of the WSX-1/TCCR subunit and the gp130 subunit) is inherently a signal-transducing receptor (see, for exemplary purposes only, Sprecher *et al.*, 1998 Biochem and Biophys Res Comm 246:82-90, specifically at p. 89, col 1, first full paragraph, incorporated by reference by Timans *et al.*, at p.20:0218). Timans *et al.*, teach that antagonizing of any of the components in the receptor subunit:ligand complex should have a beneficial effect in inflammatory diseases (p. 15, col 2:0161).

The level of skill of those in the art is high because of the complex nature of immunology and the regulation of the immune system in any given species is inherently unpredictable. The singular working model of the instant application teaches methods of assessing the role of IL-27/WSX-1 in the

development and regulation of resistance to *Toxoplasma gondii* (see p. 111). To facilitate this, WSX-1 -/- mice are used. No guidance is provided with regard to whether suppression or activation of WSX-1 will treat, prevent, or modulate any "immune disorder" or hypersensitivity disorder response in any species.

Applicants' claims are excessively broad due, in part, to the complex and diverse nature of immune disorders, hypersensitivity reactions, T-helper cell responses, the nature of the cytokine network, and infections. Applicant's claims are drawn to methods of treating, preventing, or modulating an immune disorder, T-helper cell mediated response, interferon-γ mediated immune response, or hyperactivity disorder in a patient by administering an agonist or antagonist which suppresses or enhances WSX-1 activity, thereby suppressing or activating the immune system of the patient. As stated *supra*, the specification does not reasonably provide enablement for prevention of any immune disorder in any species by any means. The skilled artisan cannot envision the prevention of an immune disorder. Prevention involves "attacking" the underlying cause of disease; i.e., disrupting the mechanisms which give rise to any given immune disorder. Due to the large quantity of experimentation necessary to determine the etiology of any the claimed immune disorders such that it can be determined whether they can be prevented, the lack of direction/guidance presented in the specification regarding same, the absence of sufficient working examples directed to same, the complex nature of the invention, the state of the prior art establishing that the etiology of immune disorders is unknown, and the breadth of the claims, undue experimentation would be required of the skilled artisan to make and/or use the claimed invention in its full scope.

Applicant has failed to provide any guidance as to the applicability of IL-27/WSX-1 to any of the recited conditions other than *Toxoplasma gondii* and *T. muris* infections in a species other than. As such, it would require undue experimentation to determine whether an agonist or antagonist of WSX-1 may be used to treat, prevent, or modulate any of the claimed "immune disorders," immune responses, or hypersensitivity disorders.

Additionally, Applicant has provided a list of disorders that Applicant claims to be "immune disorders" (see, i.e. claims 21, 23, and 73). However, the inclusion of specific non-immune conditions in these Markush groups are repugnant to one of ordinary skill in the art. The non-immune-related conditions asserted to be "immune disorders" by Applicant include, but are not limited to: alcoholic cirrhosis, biliary cirrhosis, anorexia, cancer, tumor metastasis, clostridium-associated illnesses, clostridium-associated diarrhea, a coronary condition, a coronary indication, congestive heart failure, myocardial infarction, myocardial dysfunction, a coronary artery bypass graft-associated condition, type II (adult-onset) diabetes, insulin resistance, analgesia, ischemia, cerebral ischemia, learning impairments,

neurotoxicity, ocular diseases and conditions, ocular degeneration, pain, cancer-related pain, periodontal disease, Pityriasis rubra pilaris (PRP), pre-term labor, prostatitis, prostatitis-related conditions, and side effects from radiation therapy.

Further, Applicant has failed to provide any guidance on what constitutes an active fragment of IL-27 (which is comprised of a heterodimeric subunit of p28 and EBI3). Undue experimentation would be required to determine which portions of the heterodimeric subunit may be fragmented and retain activity. Applicant has provided no guidance whatsoever on how to do this in the instant disclosure. Additionally, Applicant has failed to provide any guidance on agonistic antibodies to IL-27R/WSX-1, inactive IL-27 fragments which retains IL-27/WSX-1 binding activity, or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity.

Therefore, based on the discussions above concerning the art's recognition that, the specification fails to teach the skilled artisan how to use the claimed methods without resorting to undue experimentation to determine how to preventing or treating an immune disorder, modulate a T-helper cell mediated immune response, modulate an interferon-γ mediated immune response, treat immune hyperreactivity in a patient, or treat an immune hyperreactivity disorder in a patient by administering an antagonist or an agonist to suppress or activate the immune system of the patient.

Due to the large quantity of experimentation necessary to determine which subpopulations of patients would respond to immune suppression or immune activation or to determine whether the claimed immune disorders, hypersensitivity disorders, or immune conditions are related to WSX-1 involvement in the disease process, the lack of direction/guidance presented in the specification regarding same, the absence of sufficient working examples directed to same, the complex nature of the invention, the state of the prior art establishing that antagonizing of any of the components in the receptor subunit:ligand complex (IL-27/IL-27R , alternatively known as the complex of p28/EBI3/WSX-1/gp130) should have a beneficial effect in inflammatory diseases, and the breadth of the claims which fail to recite any correlation of patient populations with specific diseases or how to use the mutually exclusive components of the invention to agonize or antagonize the WSX-1 subunit to activate or suppress an immune response in a patient, undue experimentation would be required of the skilled artisan to make and/or use the claimed invention in its full scope.

Claim Rejections - 35 USC § 112, First Paragraph

Written Description

15. Claims 1-26 and 73 are rejected under 35 U.S.C. 112, first paragraph, as containing subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors, at the time the application was filed, had possession of the claimed invention. This is a written description rejection, rather than an enablement rejection under 35 U.S.C. 112, first paragraph. Applicant is directed to the Guidelines for the Examination of Patent Applications Under the 35 U.S.C. 112, 1 "Written Description" Requirement, Federal Register, Vol. 66, No. 4, pages 1099-1111, Friday January 5, 2001.

The claims recite a method of preventing or treating an immune disorder in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said treating or preventing comprises immune suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the recited group; wherein said treating or preventing comprises immune activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27R/WSX-1 binding affinity or an antagonist antibody that suppresses IL-27R/WSX-1 activity; a method for modulating a T-helper cell mediated immune response in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL-27, and an agonistic antibody to IL-27R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27/WSX-1 binding activity or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; wherein the T-helper cell is Th1; wherein the T-helper cell is Th2; a method for modulating an interferon-γ mediated immune response in a patient in need thereof which comprises administering to a patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL027, and an agonistic antibody to IL027R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an active IL27 fragment which retains IL-27R/WSX-1 binding affinity, or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; a method for treating immune hyperreactivity in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; a method for

treating an immune hyperreactivity disorder in a patient in need thereof, which comprises administering to said patient an effective amount of a IL-27R/WSX-1 ligand; wherein said immune disorder is selected from the group consisting of autoimmune disorders, hypersensitivity disorders, allergies, and asthma; wherein said immune disorder is selected from the recited group; a method for treating a T-helper cell mediated disorder in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein the T-helper cell mediated disorder is selected from the recited group; a method for modulating a T-helper cell mediated autoimmune response in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said T-helper cell is Th1; wherein said T-helper cell is Th2; the method of claim1 wherein the disorder is selected from the recited group.

Vas-Cath Inc. V. Mahurkar, 19 USPQ2d 1111, states that Applicant must convey with reasonable clarity to those skilled in the art that, as of the filing date sought, he or she was in possession of the invention. The invention, for purposes of the written description inquiry, is whatever is now claimed (see page 1117). A review of the language of the claim indicates that these claims are drawn to a genus, i.e., a method of preventing or treating an immune disorder, a method of modulating a T-helper cell mediated immune response, a method for modulating an interferon-γ mediated immune response, a method for treating immune hyperreactivity in a patient, or a method for treating an immune hyperreactivity disorder in a patient by administering an antagonist or an agonist to suppress or activate the immune system of the patient, wherein the patient is of any species.

To provide adequate written description and evidence of possession of a claimed genus, the specification must provide sufficient distinguishing characteristics of the genus. The factors to be considered include disclosure of complete or partial structure, physical and/or chemical properties, functional characteristics, structure/function correlation, methods of making the claimed product, or any combination thereof.

A description of a genus may be achieved by means of a recitation of a representative number of species falling within the scope of the genus or of a recitation of structural features common to the members of the genus, which features constitute a substantial portion of the genus. *Regents of the University of California v. Eli Lilly & Co.*, 119 F3d 1559, 1569, 43 USPQ2d 1398, 1406 (Fed. Cir. 1997). In *Regents of the University of California v. Eli Lilly* (43 USPQ2d 1398-1412), the court held that a generic statement which defines a genus of nucleic acids by only their functional activity does not provide an adequate written description of the genus. The court indicated that, while applicants are not required to disclose every species encompassed by a genus, the description of the genus is achieved by the

recitation of a representative number of species falling within the scope of the claimed genus. At section B(1), the court states, "An adequate written description of a DNA ... requires a precise definition, such as by structure, formula, chemical name, or physical properties, not a mere wish or plan for obtaining the claimed chemical invention."

There is a single species of the claimed genus disclosed that is within the scope of the claimed genus, *i.e.* a method of assessing the role of IL-27/WSX-1 *in vitro,* in the development and regulation of resistance to *Toxoplasma gondii* in WSX-1 deficient mice. The disclosure of a single disclosed species may provide an adequate written description of a genus when the species disclosed is representative of the genus. However, the present claim encompasses numerous species that are not further described.

The specification does not adequately describe a sufficient number of agonists or antagonists of the WSX-1 IL-27R subunit such that the same may be used to treat, prevent, or modulate any of the claimed "immune disorders," immune responses, or hypersenstitivity disorders, nor does it describe a sufficient number of possible agonists/antagonists in other species and in a way that a person of ordinary skill in the art would know that they were in possession of Applicant's invention.

Moreover, Applicant claims a method comprising immune suppression and immune activation using a IL-27R/WSX-1 ligand as either an agonist or antagonist. The use of a ligand as either an agonist or antagonist is mutually exclusive as to the function of the ligand. Further, the patient population to be treated appears to be identical. There disclosure lacks an adequate description of how one of ordinary of skill is supposed to treat the same patient population with a ligand that can be either agonistic or antagonistic. No parameters are provided such that distinct subpopulations requiring either the agonist or antagonist may be determined.

Additionally, no active fragments of IL-27 (which is comprised of a heterodimeric subunit of p28 and EBI3) are described such that the skilled artisan would be able to determine which portions of the heterodimeric subunit may be fragmented and still retain activity. Applicant has failed to provide an adequate description of agonistic antibodies to IL-27R/WSX-1, inactive IL-27 fragments which retain IL-27/WSX-1 binding activity, or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity.

In the absence of sufficient recitation of distinguishing characteristics, the specification does not provide adequate written description of the claimed genus, which is a therapeutic agent, a reference molecule, and a therapeutic index. One of skill in the art would not recognize from the disclosure that the applicant was in possession of the genus. The specification does not clearly allow persons of ordinary skill in the art to recognize that he or she invented what is claimed (see *Vas-Cath* at page 1116).

Applicant is reminded that *Vas-Cath* makes clear that the written description provision of 35 U.S.C. 112 is severable from its enablement provision (see page 1115).

Claim Rejections - 35 USC § 112, Second Paragraph

16. The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

17. Claim 1-26 and 73 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. Applicant claims a method comprising immune suppression and immune activation using a IL-27R/WSX-1 ligand as either an agonist or antagonist. The use of a ligand as either an agonist or antagonist is mutually exclusive as to the function of the ligand. Additionally, the patient population to be treated appears to be identical. It is unclear from the disclosure how one of skill in the art is supposed to treat the same patient population with a ligand that can be either agonistic or antagonistic. No parameters are provided such that distinct subpopulations requiring either the agonist or antagonist may be determined.

18. Claims 1-26 and 73 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention. The specification refers to the ligand of interest as IL-27/WSX-1 throughout most of the specification and all of the examples. The ligand of interest is recited as IL-27R/WSX-1 in one paragraph on page 1 of the disclosure, but is primarily identified as IL-27/WSX-1 throughout the remainder of the disclosure. However, the claims recite the ligand of interest as IL-27R/WSX-1. IL-27 is considered the ligand of the IL-27 receptor (IL-27R). WSX-1 is known to be one of the two subunits of the IL-27R, the other being gp130. For purposes of compact prosecution, the designations of either IL-27/WSX-1 or IL-27R/WSX-1 will be read as referring to the WSX-1 portion of the IL-27R subunit of the IL-27 ligand/receptor complex.

Claim Rejections - 35 USC § 102

19. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless –

(a) the invention was known or used by others in this country, or patented or described in a printed publication in this or a foreign country, before the invention thereof by the applicant for a patent.

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e) the invention was described in (1) an application for patent, published under section 122(b), by another filed in the United States before the invention by the applicant for patent or (2) a patent granted on an application for patent by another filed in the United States before the invention by the applicant for patent, except that an international application filed under the treaty defined in section 351(a) shall have the effects for purposes of this subsection of an application filed in the United States only if the international application designated the United States and was published under Article 21(2) of such treaty in the English language.

(f) he did not himself invent the subject matter sought to be patented.

20. Claims 1-26 and 73 are rejected under 35 U.S.C. 102(a) as being anticipated by Timans *et al.*, US Patent Application Publication 2002/0164609 A1 (publication date 7 November 2002).

The claims recite a method of preventing or treating an immune disorder in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said treating or preventing comprises immune suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the recited group; wherein said treating or preventing comprises immune activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27R/WSX-1 binding affinity or an antagonist antibody that suppresses IL-27R/WSX-1 activity; a method for modulating a T-helper cell mediated immune response in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL-27, and an agonistic antibody to IL-27R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27/WSX-1 binding activity or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; wherein the T-helper cell is Th1; wherein the T-helper cell is Th2; a method for modulating an interferon-γ mediated immune response in a patient in need thereof which comprises administering to a patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL027, and an agonistic antibody to IL027R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an active IL27 fragment which retains

IL-27R/WSX-1 binding affinity, or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; a method for treating immune hyperreactivity in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; a method for treating an immune hyperreactivity disorder in a patient in need thereof, which comprises administering to said patient an effective amount of a IL-27R/WSX-1 ligand; wherein said immune disorder is selected from the group consisting of autoimmune disorders, hypersensitivity disorders, allergies, and asthma; wherein said immune disorder is selected from the recited group; a method for treating a T-helper cell mediated disorder in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein the T-helper cell mediated disorder is selected from the recited group; a method for modulating a T-helper cell mediated autoimmune response in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said T-helper cell is Th1; wherein said T-helper cell is Th2; the method of claim1 wherein the disorder is selected from the recited group.

Timans *et al.*, teach an interleukin subunit named IL-D80, also known as p28 (p. 2, col 1, paragraph 0014). The heterodimer IL-D80/EBI3 composite cytokine is also known as IL-27 (p. 2, col 1, paragraph 0014). One of the subunits comprising the IL-27 receptor is WSX-1 (also known as TCCR or WSX-1/TCCR) (p. 2, col 1, paragraph 0014 and p. 15, col 1, paragraph 0159). WSX-1/TCCR is one of the two IL-27 receptor subunits (p. 15, col 2, paragraph 0161), the other being gp130. Timans *et al.*, teach methods for modulating or intervening in the immune response of subjects with "inflammation, including, but not limited to ulcerative colitis, arthritis, etc." (p. 3, col 1, paragraph 0027). "EBI3 [one half of the IL-27 heterodimer] has been shown to be upregulated in colonic tissue of patients suffering from gut inflammation disorders, e.g., ulcerative colitis, suggesting that the composite cytokine [p28] may also be involved" (p. 15, col 2, paragraph 0160).

Timans *et al.*, also teach the administration of an agonist or antagonist of IL-D80 [p28], IL-27, or WSX-1/TCCR, "in the treatment of abnormal medical conditions, including immune disorders, e.g...inflammation..." (p. 4, col 1, paragraph 0039), those agonists or antagonists include functional or receptor [WSX/TCCR] antagonists (p. 4, col 1, paragraph 0039), and agonists or antagonists where the binding component is an Fv, Fab, or Fab2 fragment (p. 2, col 2, paragraph 0019). Additionally, Timans *et al.*, teach the therapeutic use of blocking antibodies to IL-D80 (also called p28 and TEASRL), IL-27, or WSX-1/TCCR as antagonists and of stimulatory antibodies as agonists (p. 12, col 2, paragraph 0135). Further, Timans *et al.*, teach the role for the composite cytokine [p28/EBI3] and its associated receptor subunit WSX-1/TCCR in inflammatory responses, by describing how antagonizing the function of any of

the components in the receptor subunit:ligand complex should have a beneficial effect in inflammatory diseases, e.g., inflammatory bowel disease, rheumatoid arthritis, etc. (p. 15, col 2, paragraph 0161).

Further, antibodies to p28, EBI3, WSX-1/TCCR, and gp130 were commercially available at the time the instant application was filed (see, Timans et al., 2002/0164609 A1, specification p. 23, paragraph 0068). It is well known in the art that the heterodimeric IL-27 receptor (comprised of the WSX-1/TCCR subunit and the gp130 subunit) is inherently a signal-transducing receptor (see, for exemplary purposes only, Sprecher *et al.*, 1998 Biochem and Biophys Res Comm 246:82-90, specifically at p. 89, col 1, first full paragraph, incorporated by reference by Timans *et al.*, at p.20, paragraph 0218). Timans *et al.*, teach that antagonizing of any of the components in the receptor subunit:ligand complex should have a beneficial effect in inflammatory diseases (p. 15, col 2, paragraph 0161).

Timans *et al.*, teach a binding composition that includes a Fv, Fab, or Fab2 fragment (p. 2, col 2, paragraph 0019). Timans *et al.*, also teach a wide variety of detectable labels and conjugation techniques at p8, paragraph 0092. Further, Timans *et al.*, teach nucleic acids encoding an IL-D80 (p28) polypeptide or fusion peptide (p 2, paragraph 0020) that can be used as antagonists (see also, Timans et al., at p.9, paragraphs 0096-0110).

Timans *et al.*, teach stimulatory antibodies as agonists (p. 12, col 2, paragraph 0135). Timans *et al.*, also teach antibodies to p28 as antagonists that inhibit functional binding (p.8, paragraph 0089). Timans *et al.*, explain that these antagonists can be useful as non-neutralizing antibodies and can be coupled to toxins or radionucleotides so that when the antibody binds to the antigen, a cell expressing it, e.g., on its surface, is killed (p. 8, paragraph 0089). Decreased p28 expression would occur if a cell were killed as a result of this antibody-dependent cytotoxic killing. Timans *et al.*, also teach antibodies to p28 for stericly blocking binding to a receptor (p. 7, paragraph 0083) and soluble receptors at p. 15, paragraph 0159. Timans *et al.*, teach each and every limitation of the claims in the instant application.

21. Claims 6-17 and 24-26 are rejected under 35 U.S.C. 102(a) as being anticipated by Villarino *et al.*, (Immunity. 2003 Nov; 19(5):645-55).

The claims recite a method for modulating a T-helper cell mediated immune response in a patient in need thereof, which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL-27, and an agonistic antibody to IL-27R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an inactive IL-27 fragment which retains IL-27/WSX-1 binding activity or an antagonist antibody to IL-27R/WSX-1 which

suppresses IL-27R/WSX-1 activity; wherein the T-helper cell is Th1; wherein the T-helper cell is Th2; a method for modulating an interferon-γ mediated immune response in a patient in need thereof which comprises administering to a patient an effective amount of an IL-27R/WSX-1 ligand; wherein said modulation is suppression and said ligand is an IL-27R/WSX-1 agonist; wherein said agonist is selected from the group consisting of IL-27, an active fragment of IL027, and an agonistic antibody to IL027R/WSX-1 which enhances IL-27R/WSX-1 activity; wherein said modulation is activation and said ligand is an IL-27R/WSX-1 antagonist; wherein said antagonist is an active IL27 fragment which retains IL-27R/WSX-1 binding affinity, or an antagonist antibody to IL-27R/WSX-1 which suppresses IL-27R/WSX-1 activity; a method for modulating a T-helper cell mediated autoimmune response in a patient in need thereof which comprises administering to said patient an effective amount of an IL-27R/WSX-1 ligand; wherein said T-helper cell is Th1; wherein said T-helper cell is Th2.

Villarino et al., teach methods of modulating T-helper cell immune responses in WSX-1 -/- mice and wild type controls by infection with *Toxmoplasma gondii* (p. 645, Summary). Villarino et al., showed increased cytokine production in WSX-1 -/- mice including IFN-γ and IL-12 levels, post infection (p. 646, column 2, third paragraph). Assessment of the T-helper cell response post-infection showed an increase in $CD25^{high}/CD62L^{low}$ CD4+ T-cells in WSX-1 -/- mice, even after BrdU administration and subsequence incorporation (which decreased the activated CD4+ levels in wild-type mice) (p. 649, column 1, first full paragraph). Villarino et al., show that while WSX-1 is not necessary for the generation of highly activated Th1 effector T-cells following challenge with *T. gondii*, WSX-1 is required to regulate the intensity and duration of infefction induced Th1 responses (p. 649, column 2, fist paragraph). RT-PCR showed increased expression of IL-12p28 and EBI3 mRNA indicating the presence of IL-27, a WSX-1 ligand (p. 650, column 1, first full paragraph). Villarino et al., also teach that WSX-1 is crucial for the optimal production of IFN-γ by naïve CD4+ T-cells that have been activated under nonpolar conditions (p. 650, column 2, first paragraph; Figure 6). Villarino et al., also teach that antigen dose and cytokine environment are critical factors in the differentiation of naïve CD4+ T-cells into effector Th1 and Th2 cells (p. 651, column 2, third paragraph).

22. Claims 1-26 and 73 are rejected under 35 U.S.C. 102(b) as being anticipated by De Sauvage et al., WO 01/29070 (26 April 2001) (see also US Patent Application Publication 2004/0234522 A1).

The claims recite as stated *supra*.

DeSauvage et al., teach methods for the treatment and diagnosis of immune related diseases including those mediated by cytokines released primarily by either Th1 or Th2 cells in response to

antigenic stimulation, methods for biasing the differentiation of T-cells in either Th1 or Th2 subtypes, based on the relative expression levels of TCCR (WSX-1) and its agonists or antagonists, and methods of diagnosing Th1- and Th2-mediated diseases (abstract). Anti-TCCR (WSX-1) antibodies, including agonist antibodies and antagonist antibodies, are taught at p. 3, last paragraph and pp. 51-56. Antagonists of a TCCR (WSX-1) that inhibit one or more functions of the TCCR (WSX-1) polypeptide and TCCR (WSX-1) agonists that stimulate or enhance the activities of the TCCR (WSX-1) polypeptide are taught on page 4. Immune related diseases are defined on p. 8 as a disease in which a component of the immune system in a mammal caused, mediates, or otherwise contributes to a morbidity in a mammal. Immune-mediated inflammatory diseases, non-immune mediated inflammatory diseases, infectious diseases, immunodeficiency diseases, and neoplasia are included in this definition (see, p. 8) and exemplified by disease name on p. 9. Uses of TCCR (WSX-1) are taught, beginning on p. 35. Immunostimulating components comprising TCCR (WSX-1) polypeptides used as therapeutics are taught at pp. 45 and 47. Identification of agonists and antagonists of TCCR (WSX-1) are taught at pp. 48-49. Pharmaceutical compositions comprising TCCR (WSX-1) are taught at p. 58-59. Methods of treatment using TCCR (WSX-1) polypeptides, antibodies, and other active compounds to treat various immune related diseases and conditions such as T-cell mediated diseases, including those characterized by infiltration of inflammatory cells into a tissue, stimulation of T-cell proliferation, inhibition of T-cell proliferation, increased or decreased vascular permeability or the inhibition thereof, are taught, along with exemplary disorders and conditions on pp. 59-63. See also, Example 12, pp. 79-81.

23. Claims 1-26 and 73 are rejected under 35 U.S.C. 102(b) as being anticipated by Bennett et al., WO 97/25425 (17 July 1997).

The claims recite as stated *supra*.

Bennett et al., teach methods of using the WSX-1 ligands (abstract), WSX-1 fusion proteins (p. 4) and anti-WSX-1 receptor agonist and antagonist (neutralizing) antibodies (pp. 4-5) for the treatment of hematopoietic disorders such as leukemia, lymphoma, and anemia and for enhancement of lymphopoiesis in disorders such as HIV/AIDS and infections (p. 6). Treatment of malignancies are taught at p. 7 and treatment of metabolic disorders are taught on p. 42. Additional therapeutic uses for the WSX-1 receptor are taught on p. 41. Therapeutic uses for WSX-1 receptor ligands and antibodies are taught on pp. 56-59.

24. Claims 1-26 and 73 are rejected under 35 U.S.C. 102(e) as being anticipated by Timans *et al.*, US 2002/0164609 A1 (publication date 7 November 2002).

The claims recite as stated *supra*. Timans *et al.*, teach as stated *supra*.

25. Claims 1-26 and 73 are rejected under 35 U.S.C. 102(e) as being anticipated by Matthews *et al.*, US Patent 7,074,397 B1 (11 July 2006, benefit to 8 January 1996).

The claims recite as stated *supra*.

Matthews et al., teach uses for WSX ligands, anti-WSX-1 receptor antibodies or the obesity protein (column 1, lines 24-35), the WSX receptor or WSX ligand antibodies (column 4, lines 38-40) in the treatment of hematopoietic disorders such as leukemia, lymphoma, anemia, and for enhancement of lymphopoiesis in disorders such as HIV/AIDS, infections, and malignancies (column 4, lines 43-54). Antagonists of WSX receptor activation (WSX receptor extracellular domain, WSX receptor immunoadhesins, WSX receptor antisense nucleic acids, and WSX neutralizing antibodies) used to treat a pathological disorder are taught at column 41, lines 21-29. Pharmaceutical compositions comprising the WSX receptor are taught at column 41, lines 43-47. Additional therapeutic uses for the WSX-1 receptor are taught at column 50, lines 45-67 to column 51, lines 1-31.

Conclusion

NO CLAIM IS ALLOWED.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Cherie M. Woodward whose telephone number is (571) 272-3329. The examiner can normally be reached on Monday - Thursday 9:00am-7:30pm (EST).

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Brenda Brumback can be reached on (571) 272-0961. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

CMW/AU 1647

Marianne P. Allen

MARIANNE P. ALLEN
PRIMARY EXAMINER
AU1647

11/24/06